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# The Effect of Birth Spacing on Child Mortality in Sweden, 1878–1926

JOSEPH MOLITORIS

SHORTER BIRTH INTERVALS have been identified as having potentially serious implications for the health of both children and their mothers (see Conde-Agudelo 2012; Kozuki et al. 2013 for recent reviews). Evidence has consistently shown that children born following shorter intervals generally have substantially higher rates of neonatal, post-neonatal, and childhood mortality than those born following longer intervals.<sup>1</sup> The overwhelming consistency of this relationship has clearly been of interest to policy makers (WHO 2007), as marginal increases in median birth intervals could lead to significant improvements in public health.

This study has three objectives. First, I examine within-family variation in the association between preceding birth intervals and infant and child mortality risks of index children to remove the potential for the relationship to be driven purely by compositional differences across mothers. This approach has rarely been adopted in the literature, yet is crucial to accurately identifying the mechanisms responsible. Second, I investigate the relationship between birth spacing and infant and child mortality using historical data (see Knodel and Hermalin 1984; Nault, Desjardins, and Légaré 1990; Pebley, Hermalin, and Knodel 1991); mine is the first study to examine how this relationship evolved over a roughly 50-year period of major economic and demographic changes. This is a significant advantage as historical populations tended to have birth spacing patterns similar to those in contemporary developing contexts, yet also had greater numbers of deaths at young ages, which can greatly improve the power of statistical analyses and aid in the search for causal mechanisms. Finally, I use variation in the timing of the previous sibling's death to isolate the role of the various causal mechanisms. Most studies have only treated the survival status of previously born children as a binary variable (i.e. alive or dead). This study, in contrast, will examine how the death of the previous sibling influenced the relationship between birth intervals and the index child's mortality depending on whether that death occurred in the pre-gestation, gestation, or postnatal period of the index child. This innovation can help us better understand the mechanisms at play, as certain causal mechanisms should

only operate in the prenatal period while others primarily operate in the postnatal period.

## Birth spacing and mortality

The causal mechanisms linking birth interval length to mortality fall into four broad categories: maternal depletion, sibling competition, infection transmission, and family frailty. The maternal depletion hypothesis argues that short intervals do not allow mothers adequate time to recover their nutritional stores, leading to suboptimal nutritional status during pregnancy. This, in turn, leads to compromised fetal growth and increased risks of adverse maternal and perinatal outcomes (Winkvist, Rasmussen, and Habicht 1992). Related hypotheses argue that short intervals do not allow women's bodies to fully recover from their previous birth in other ways, such as by depleting folate stores or by decreasing muscle tone in reproductive tissues (Conde-Agudelo et al. 2012; Smits and Essed 2001).

Others have argued that sibling competition is the primary factor leading to a greater risk of mortality: the closer siblings are in age, the more likely that they will demand the same parental resources. Here resources may refer to parents' income, time, housing, or health. Competition for some resources, like breastmilk, could be especially acute as a result of short birth intervals. Findings from developing countries have shown that overlapping periods of pregnancy and breastfeeding are not uncommon during short birth intervals (Boerma and Bicego 1992), which may lead to lower quality or quantity of breastmilk for the child born at the end of the interval and diminished neonatal growth (Marquis et al. 2002; Marquis et al. 2003). The scant research on the effects of overlap between breastfeeding and pregnancy has found no association with fetal growth, however (Merchant, Martorell, and Haas 1990). At ages beyond weaning, competition for the quality and quantity of calories obtained from food would obviously be more relevant, though it would be more a function of family size than short intervals per se.

It has also been suggested that short intervals may be primarily mediated by the transmission of infectious disease in two ways: horizontally and vertically. The horizontal transmission hypothesis argues that short intervals lead to higher rates of infant and child mortality because closely spaced siblings are susceptible to diseases that can be easily transmitted from an older to a younger sibling with a limited immune system (Boerma and Bicego 1992). The vertical transmission hypothesis, on the other hand, posits that short intervals may be a result of maternal infections, which in turn are transmitted *in utero* and lead to worse perinatal outcomes. It is unclear, however, whether vertical transmission would independently influence perinatal outcomes via birth spacing or whether it would be a joint determinant of both birth interval length and the probability of dying at young ages.

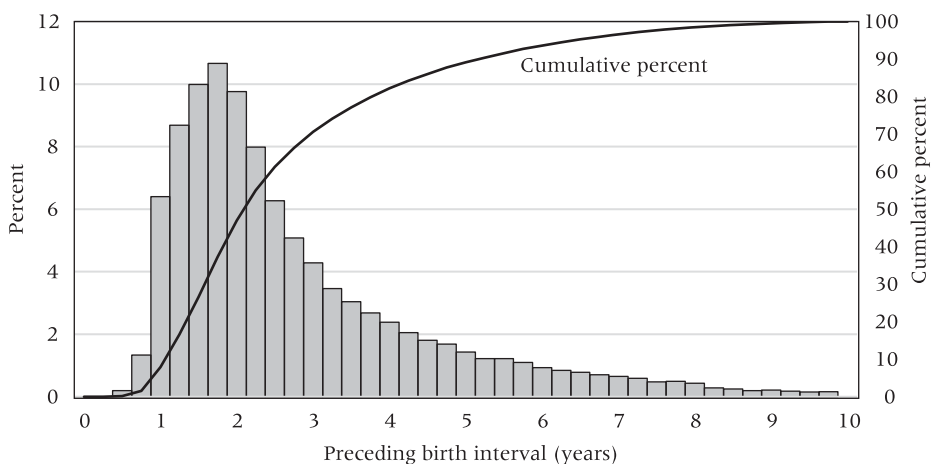
Finally, the family frailty hypothesis argues that the relationship between interval length and child mortality is spurious (Miller 1989). In other words, unobserved factors may cause women to have both short intervals and higher child mortality. One example of this would be related to maternal breastfeeding practices. Mothers who do not breastfeed will not have a period of lactational infecundability that would have reduced their exposure to pregnancy.

## Data

The data used in this study come from the Roteman Database, a longitudinal individual-level population register maintained for Stockholm, Sweden between 1878 and 1926. At the start of the coverage period, the city was divided into 16 districts of about 10,000 inhabitants each; this number grew to 36 districts by the end of period. The database includes all individuals ever living in Stockholm during the period as well as all live births, deaths, marriages, and migration and was updated annually upon census registration and at the occurrence of demographic events.<sup>2</sup> Over 70 percent of the original source material has so far been digitized. In addition to demographic information, the database has information on occupations, illegitimacy, smallpox vaccination, and location of residence within the city.

The sample in the present study was restricted to higher-order births (parity 2+) to women who had their first marital birth between 1878 and 1920.<sup>3</sup> This restriction was made for two reasons. First, it is possible that births which occurred prior to the registration period (i.e. 1878) are under-reported, particularly for children who died. Under-reporting would affect the recorded length of birth intervals preceding the birth of children born during the registration period. Second, although the data coverage is terminated in 1926, a maximum first-birth cohort of 1920 was selected in order to avoid artificially shortening the length of preceding birth intervals of individuals born toward the end of the coverage. This means that, while 1920 was the final first-birth cohort observed, their children were able to be born up until 1926. Multiple births were assigned the parity of the birth order just as any birth would be, while the parity of the birth occurring following a multiple birth depended on how many children were born in the multiple birth. So, for example, twins born at parity 2 would both be considered second born, while the birth occurring after the twins would be considered the fourth born. Multiple births were rare, however, accounting for about one percent of all higher-order births.

The distribution of preceding birth intervals is shown in Figure 1. The median interval length for these cohorts of women was 2.3 years for all parities combined and was shorter at lower parities, likely due to a combination of greater fecundability and lower levels of fertility control at these parities. The median interval length increased continuously across the

**FIGURE 1** Distribution of preceding birth intervals

NOTE: Distribution represents intervals for children born at parity 2+ between 1878 and 1926.  
 SOURCE: Roteman Database, own calculations.

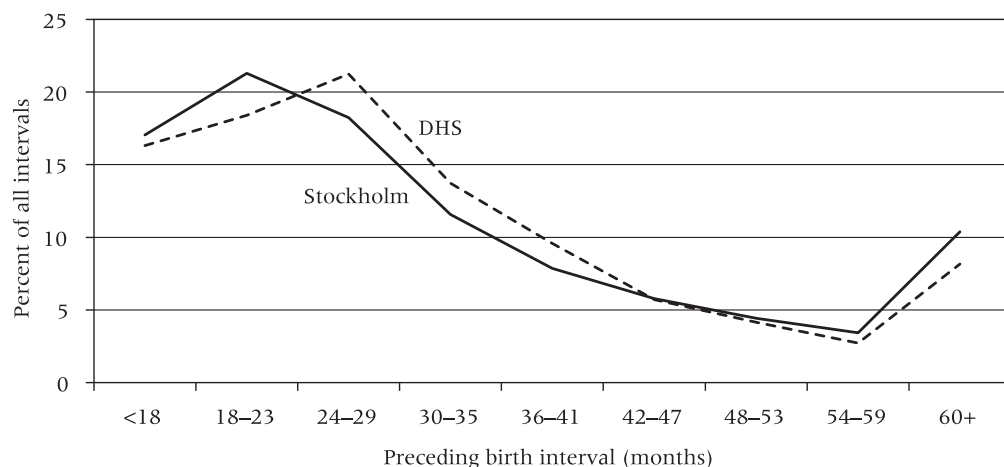
cohorts to about three years by the end of observation (Molitoris and Dribe 2016b). About 20 percent of all index children were born within 18 months of the previous birth, and just over 40 percent were born following an interval of less than two years. Intervals exceeding five years in length were rare, accounting for under 10 percent of all births.

The distribution of birth intervals in Stockholm during the late nineteenth and early twentieth centuries is remarkably similar to that found in contemporary developing countries. Figure 2 compares the distribution of birth intervals in Stockholm to a weighted average of 17 DHS samples from Asia, Africa, and South America collected between 1990 and 1997. The primary difference between the distributions is that birth intervals in Stockholm were slightly shorter. Despite being separated by time and geography, both Stockholm and many of the DHS countries were still relatively underdeveloped economically. Thus, the lessons we can learn from Stockholm's past may be relevant to contemporary developing countries.

## Methods

Ordinary least squares (OLS) is used to estimate linear probability models (LPM) of neonatal (< 28 days), post-neonatal (28–365 days), younger child (1–4 years), and older child (5–9 years) mortality. Each model is necessarily conditional on surviving until the start of the age span of interest. So, for example, to be included in the model of younger child mortality, a child must have survived and been observed until at least age one. The models' main independent variable is the length of the preceding birth interval. The variable is included as a quartic function to account for the well-known

**FIGURE 2 Comparison of distribution of birth interval length (in months) between Stockholm (1876–1926) and 17 DHS samples (1990–1997)**



NOTE: DHS figures are a weighted average of intervals based on 17 DHS samples collected between 1990 and 1997. Figures refer only to parity 2+.

SOURCE: Roteman Database, own calculations. Figures for DHS countries calculated based on data provided in Rutstein (2005).

nonlinear relationship that has been found in virtually all of the literature on the subject (see Hobcraft, McDonald, and Rutstein 1985; Rutstein 2005).

Each model controls for variables that are specific to the index child and preceding birth. The models include controls for the age of the index child's mother at birth, multiple births, sex, birth order, birth year, and month of birth, since each of these variables may influence mortality at young ages. Some of these controls, like mother's age at birth and multiple births, will also be negatively correlated with birth intervals and positively correlated with mortality. Month of birth is included to capture seasonality in early-life disease environments that may also be correlated with the length of birth intervals. The models include a dummy variable indicating whether the at-risk child was censored before reaching the end of the age span of interest in order to control for the fact that deaths of children who are censored because of migration or the ending of the ledger period will not be observed. The models also include controls pertaining to the previous birth. The first is the sex of the previously born child, as male children historically tended to have higher odds of dying and also are more likely to be born prematurely, which may influence the length of the preceding interval of the index child (Astolfi and Zonta 1999). The second is the survival status of the previous child, which has been operationalized in a way that differs from previous studies.

Typically, previous research has ambiguously defined the survival status of the previous child as a binary indicator of infant or child mortality (e.g. Cleland and Sathar 1984; Gubhaju 1985; Hobcraft, McDonald, and

Rutstein 1985; Pebley, Hermalin, and Knodel 1991; Whitworth and Stephenson 2002; Zenger 1993). This definition is reasonable if one is only interested in capturing death clustering or in proxying family frailty. But if one wishes to use sibling deaths as a way to identify other mechanisms, this definition is inadequate. Ideally, the survival status of the previous child should be identified with respect to the age of the index child. Only one study has done this and only for the postnatal period (Alam 1995). The present study adopts this approach by defining sibling survival as a categorical variable that measures the previous child's survival status as: (1) alive, (2) died before the index child's gestation (more than 280 days before the birth of the index child), (3) died during the index child's gestation (280 days to 1 day before the birth of the index child), (4) died during the neonatal period of the index child (birth to 27 days old), (5) died during the post-neonatal period of the index child (28 to 365 days old), (6) died during younger childhood of index child (1 to 4 years old), (7) died during older childhood of index child (5 to 9 years old). If the death of a previous child occurred during the ages of analysis for the index child, but *after* the death of the index child, the variable was categorized as alive to reduce the chance of reverse causality.<sup>4</sup> This variable will first be included as a control variable and later will be interacted with the length of the preceding interval to determine whether there are heterogeneous effects of child survival at different interval lengths.<sup>5</sup>

There are several advantages to defining the previous child's survival as a categorical variable. First, it allows one to use the timing of sibling deaths to investigate the role of various causal mechanisms (Table 1). Deaths that occur in the index child's prenatal period cannot influence the horizontal transmission of infection between siblings, so if the death of the previous sibling reduces the risk of mortality of the index child and this relationship is disproportionately strong after shorter intervals, it may indicate a role for either maternal depletion or sibling competition; if it increases the risk of mortality of the index child, it may suggest a role for the vertical transmission of diseases. For deaths of previous children occurring in the index child's postnatal period, the interpretation becomes less clear. If the death

**TABLE 1** Expected relationship between the death of a previous child in the prenatal and postnatal periods and the mortality risk of the index child under different mechanisms

Mechanism	Prenatal	Postnatal
Sibling competition	Negative	Negative
Maternal depletion	Negative	None
Vertical transmission of infection	Positive	None
Horizontal transmission of infection	None	Positive
Family frailty	Positive	Positive

of the preceding child reduces the index child's risk of dying, this may be taken as support for the competition hypothesis (i.e. the removal of the sibling has improved the availability of resources for the index child). An increase in the index child's risk of dying as a result of the previous child's death may indicate an effect of family frailty. Isolating the role of the horizontal transmission of infection is not as simple, as one could potentially expect either a negative or a positive association with the death of a preceding sibling. On the one hand, the death of a sibling may remove a source of infection from the home, greatly improving individual survival chances, and would therefore be negatively associated with the risk of dying; on the other hand, if individuals born after shorter intervals were more susceptible to infection, the death of a sibling may be positively associated with an increase in the risk of dying if it serves as an indication of infection. What is certain, however, is that if the death of a sibling in the index child's postnatal period increases the risk of dying, it cannot be related to sibling competition. One should emphasize that the death of an index child as a response to the death of sibling is obviously an extreme outcome. All of the proposed mechanisms will work in much subtler ways than leading to death. Nonetheless, because historical data on morbidity are difficult to find, we must rely on a cruder, but concrete, indicator of these mechanisms, such as mortality responses.

Categorizing the previous child's survival status in this way also allows one to control for short birth intervals that are themselves a product of the previous child's death. For example, some intervals may be shortened if the death of the previous child occurs during breastfeeding, which will reduce the period of lactational infecundability and increase women's risk of pregnancy. The category pertaining to the pre-gestational period can remove this confounding influence.

To control for the fact that women who have short birth intervals may differ from other women for unobservable reasons, all of the models are estimated using maternal fixed effects. This strategy takes into account some factors that have generally been ignored in the literature. First, it eliminates the potential for the relationship between interval length and mortality to be driven purely by unobserved compositional differences between short and long spacers. All models compare only those siblings who have the same mother. Second, the strategy can at least partially control for factors shared among biological siblings and their mother that may contribute to their risk of dying at young ages. Some of this risk will be attributable to the influence of family frailty. Only a handful of studies have addressed the potential bias arising from family factors (Curtis, Diamond, and McDonald 1993; Saha and van Soest 2013; Whitworth and Stephenson 2002; Zenger 1993).

The means and distribution of the models' variables are given in Table 2. Overall, the models included 107,000–123,000 individual children



**TABLE 2** Mean values of variables included in analysis

	Neonatal	Post-neonatal	Younger child	Older child
Interval (years)	2.9	3.0	3.0	3.0
<18 months	18.5	18.4	18.1	18.1
18–23	20.5	20.6	20.5	20.5
24–35	28.7	28.7	28.8	28.6
36–47	13.1	13.1	13.2	13.3
48–59	7.6	7.6	7.7	7.7
60+	11.6	11.6	11.7	11.9
Survival status of previous child (percent distribution)				
Alive	96.5	95.8	94.5	94.0
$x < -280$ days	2.4	2.4	2.3	2.2
$-280 \leq x < 0$	0.9	0.9	0.8	0.8
$0 \leq x < 28$	0.2	0.1	0.1	0.1
$28 \leq x < 365$		0.9	0.7	0.7
$1 \leq x < 5$ years			1.6	1.4
$5 \leq x < 10$				0.9
Mother's age at birth (years)	30.8	30.8	30.8	30.8
Born a twin (percent)	1.0	1.0	1.0	1.0
Sex of index child (percent male)	51.2	51.1	50.8	50.8
Previous child's sex (percent male)	50.7	50.7	50.7	50.9
Birth order	4.2	4.2	4.2	4.1
Birth year	1903.0	1903.1	1903.3	1903.8
Censored before reaching end of age span (percent of individuals at risk)	0.3	5.1	21.9	40.7
Children at risk	123,031	121,630	116,258	107,719
Deaths	1,401	5,381	5,514	1,373
Mothers	43,707	43,564	42,984	42,006

NOTE: Mortality categories defined as: neonatal (within 28 days of birth), post-neonatal (between 28 and 365 days), younger child (between ages 1 and 4), older child (between ages 5 and 9).  $x$  refers to the date of death of the previous child with respect to the age of the index child. Negative integers represent the prenatal period of the index child, positive integers the postnatal period. ( $x < -280$  days = previous child died before gestation of the index child.)

SOURCE: Roteman Database.

born to around 43,000 women, with the larger samples for mortality at younger ages. In total, nearly 13,700 infant and child deaths are observed. The mean preceding interval for index children was just under three years, though, as mentioned above, the median was considerably shorter. The mean age at birth was around 30 years, while the mean parity was about four. About 5 percent of all index children experienced the death of an older sibling, and in about half of those cases the death occurred in the prenatal period. Although the sample size became smaller for older age groups, it is clear from the summary statistics that the samples did not differ in any substantial way from one another.<sup>6</sup>

Results

Preceding birth interval length and mortality

The results of the models are presented with and without maternal fixed effects in Tables 3 and 4 respectively. There was a strong negative effect of birth intervals on both neonatal and post-neonatal mortality in both models, with the effect size significantly larger for post-neonatal and younger child mortality risks than for neonatal mortality. For children beyond age five years, there was no significant association between interval length and mortality, all else being equal. That the relationship between birth intervals and mortality is stronger for the post-neonatal period is consistent with previous research (Cleland and Sathar 1984; Hobcraft, McDonald, and Rutstein 1985; Millman and Cooksey 1987; Whitworth and Stephenson 2002), although some studies have found neonatal mortality to be more sensitive to short preceding intervals (Boerma and Bicego 1992; DaVanzo et al. 2008).

TABLE 3 OLS estimates of the effects of birth intervals on infant and child mortality

	Neonatal	Post-neonatal	Younger child	Older child
Interval	-1.8***	-3.3***	-1.8***	-0.5
Survival status of previous child				
Alive	(ref)	(ref)	(ref)	(ref)
$x < -280$ days	17.8***	51.5***	37.6***	-1.5
$-280 \leq x < 0$	22.1***	58.7***	44.5***	5.3
$0 \leq x < 28$	330.6***	125.4***	51.8	-13.7***
$28 \leq x < 365$		186.5***	67.8***	1.8
$1 \leq x < 5$ years			163.8***	10.3***
$5 \leq x < 10$				42.0***
Mother's age at birth	0.2***	0.5***	0.2*	0.0
Born a twin	8.1	-1.9	9.5	4.2
Sex of index child	3.3***	8.7***	4.0***	0.9
Previous child's sex	0.9	2.0	-0.4	-1.2**
Birth order	1.2***	4.5***	5.3***	0.6***
Birth year	-0.1	-1.1***	-4.3***	-1.2***
Birth year <sup>2</sup>	0.0	0.0*	0.1***	0.0***
Censored before reaching end of age span	-12.6***	-40.6***	-56.3***	-21.6***
Constant	-0.6	33.3***	93.3***	37.2***
R <sup>2</sup>	0.02	0.021	0.03	0.004
N	122,622	115,445	90,727	63,903

\*p<0.1; \*\*p<0.05; \*\*\*p<0.01.  
NOTE: For mortality categories see note to Table 2. Coefficients multiplied by 1000 to facilitate interpretation.  
SOURCE: Roteman Database.

**TABLE 4 Maternal fixed-effect estimates of the effects of birth intervals on infant and child mortality**

	Neonatal	Post-neonatal	Younger child	Older child
Interval	−3.0***	−6.7***	−6.6***	0.0
Survival status of previous child				
Alive	(ref)	(ref)	(ref)	(ref)
$x < -280$ days	−24.7***	−10.1***	−6.4***	−1.5***
$-280 \leq x < 0$	11.4**	−3.9***	−13.4***	−1.2***
$0 \leq x < 28$	230.7***	9.8***	−13.6***	−2.6
$28 \leq x < 365$		12.5***	−14.5***	−2.1***
$1 \leq x < 5$ years			2.5***	−10.5***
$5 \leq x < 10$				−8.5***
Mother's age at birth	0.4***	1.1***	0.3*	−0.2
Born a twin	12.1*	16.0**	11.9	7.7
Sex	3.6***	7.6***	3.5**	0.2
Previous child's sex	1.3	2.2*	1.9	−1.0
Birth order	−0.3	−0.4	1.5**	−0.3
Birth year	1.5***	6.8***	3.5***	0.1
Birth year <sup>2</sup>	0.0	−0.1***	0.0	0.0***
Censored before reaching end of age span	−16.4***	−52.6***	−72.0***	−25.0***
R <sup>2</sup>	0.012	0.019	0.027	0.023
N	123,031	121,630	116,258	107,719

\*p&lt;0.1; \*\*p&lt;0.05; \*\*\*p&lt;0.01.

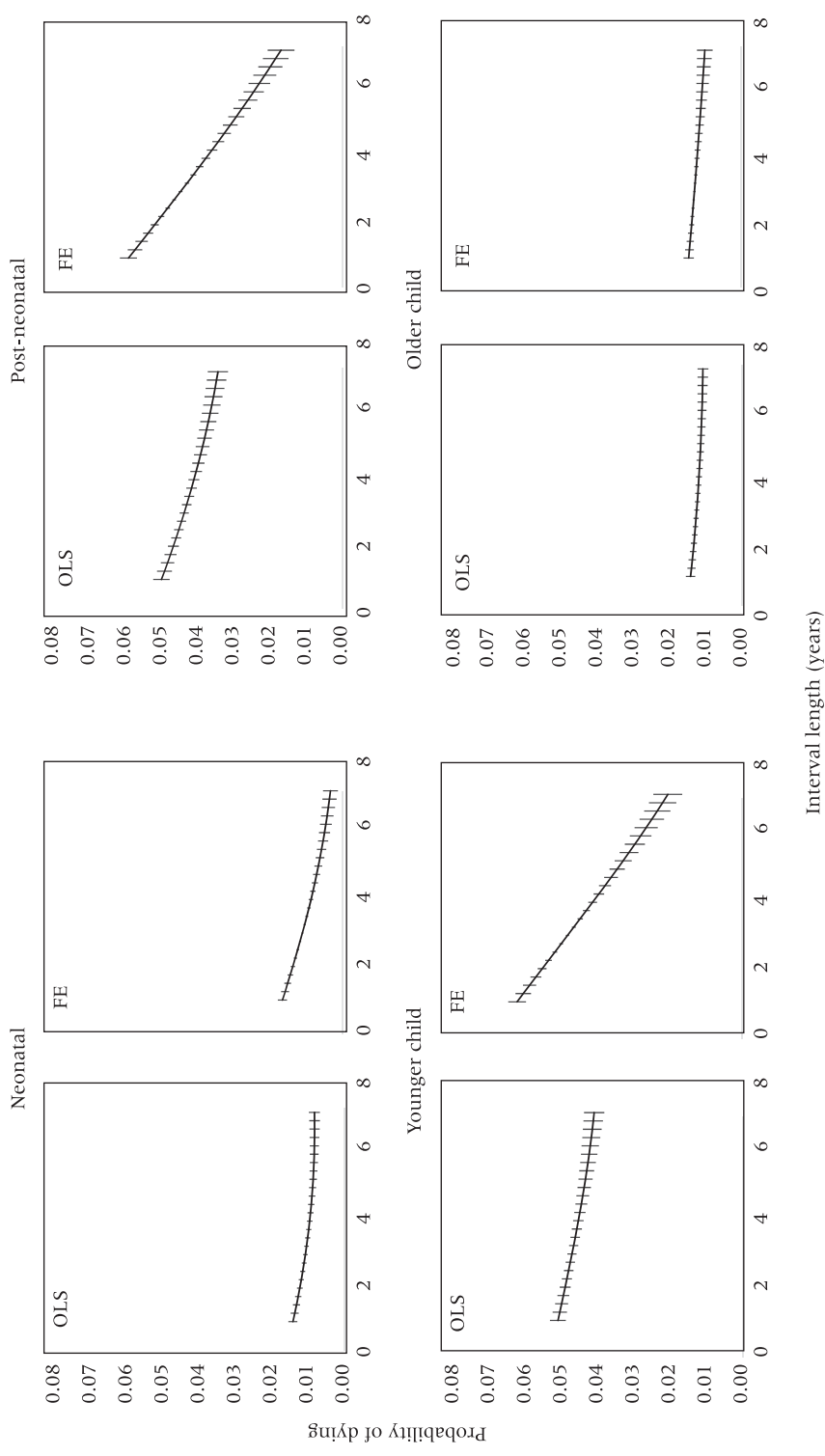
NOTE: For mortality categories see note to Table 2. Coefficients multiplied by 1000 facilitate interpretation.

SOURCE: Roteman Database.

To examine how interval length influenced the probability of dying, predicted probabilities were generated from both the OLS and fixed-effects models (Figure 3). A few features are worth noting. First is the shape of mortality risk across different interval lengths. Studies using cross-sectional data have repeatedly found that the relationship between interval length and mortality risks is nonlinear (see Rutstein 2005), with risks being especially high for intervals of less than two years, declining for intervals of 24–36 months, and then increasing again. No such pattern can be identified for this population using longitudinal data. Instead, the relationship is virtually linear. This is a significant finding, because public health recommendations have generally been made under the expectation of a plateauing and subsequent increase in infant mortality risks at interval lengths beyond certain thresholds (e.g. WHO 2007).

Another interesting finding is that the practical significance of birth interval length on mortality for post-neonates and young children changes substantially once maternal characteristics are controlled for the fixed-effects models. The probabilities predicted by the OLS model changed little as interval lengths increased. Those predicted by the fixed-effects

**FIGURE 3 Predicted probabilities of death of index child at specified ages by length of preceding birth interval, derived from OLS and fixed effects (FE) models**



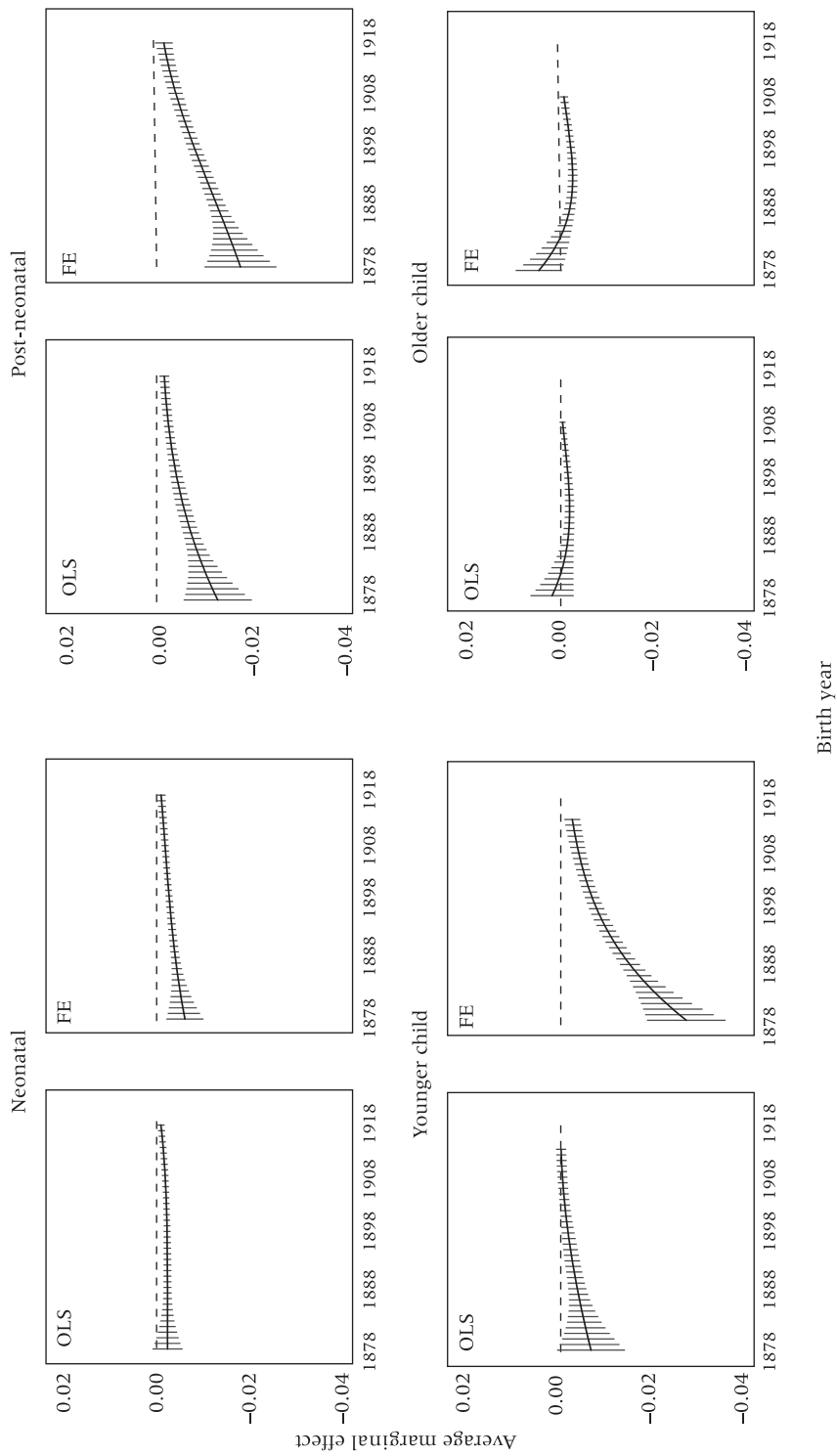
NOTE: 95 percent confidence intervals shown. Mortality categories defined as: neonatal (within 28 days of birth), post-neonatal (between 28 and 365 days), younger child (between ages 1 and 4), older child (between ages 5 and 9). FE models controlled for maternal fixed effects.  
SOURCE: Roteman database, own calculations.

models, on the other hand, indicate a strong negative relationship between the variables that was obscured by compositional differences between mothers who had different mean interval lengths. This finding also indicates that the negative effect of short birth intervals on mortality seems predominantly to affect post-neonatal and younger child mortality, both of which have a very different distribution of causes of death than neonatal mortality. In a developing-country context, infectious diseases generally account for a large share of mortality beyond the first 28 days of life, while a substantial proportion of neonatal mortality is endogenous (e.g., due to low birth weight, pre-term births, congenital defects, birth trauma). The differences in the cause-of-death distributions can point to clues regarding the relationship between interval length and mortality. If neonatal mortality is less influenced by birth intervals than mortality of older infants and children, this may suggest that mechanisms operating through disruptions in fetal growth (i.e. vertical transmission of infection, maternal depletion) may not be as important as other mechanisms.

Figure 4 shows that the association between short birth intervals and mortality weakened across birth cohorts. Only cohorts that could experience full exposure were included in these figures; for example, the final cohort in the analysis of older child mortality was born in 1916. For all age groups except older children, there was a strong negative relationship between interval length and the probability of dying early in the period of observation. When mortality was high in the earlier half of the period, there was a strong mortality-reducing effect of longer preceding intervals, especially for post-neonates and younger children. The relationship between neonatal mortality and birth interval length, though not as strong, was nonetheless negative. There was virtually no association between the mortality of older children and the length of the preceding interval at any point during the period of observation. Interestingly, the effects based on the OLS estimates suggest that interval length had almost no association with the mortality of younger children, but once maternal factors are taken into account with the fixed-effects models, a strong negative relationship emerges, as was the case for post-neonatal mortality.

Another feature of these trends is that the effects of birth intervals became substantially weaker by the end of the period. For neonatal mortality, by World War I no statistically significant effects of birth interval length could be identified. This was coincidentally also when the probability of dying before age 5 ( ${}_5q_0$ ) fell below 0.1 for the first time in Stockholm's history and the first time that Stockholm's level of child mortality was lower than the national average (Molitoris and Dribe 2016a). That the relationship disappeared over time is interesting because of what it can tell us about the mechanisms at work. For example, it seems unlikely that family frailty effects would become so much weaker at such a rapid pace. On the other hand, some of the hypothetical mechanisms are closely related to nutri-

**FIGURE 4** Average marginal effect of an increase in birth interval length on the probability of death of an index child, 1878–1926, OLS and fixed effects (FE) models



NOTE: 95 percent confidence intervals shown. Mortality categories defined as: neonatal (within 28 days of birth), post-neonatal (between 28 and 365 days), younger child (between ages 1 and 4), older child (between ages 5 and 9). FE models controlled for maternal fixed effects. Marginal effects were evaluated at the means of covariates. If confidence intervals overlap dashed line, there was no statistically significant effect at the 5 percent level.

SOURCE: Roteman database, own calculations.

tion (maternal depletion, sibling competition) while others have more to do with the disease environment (vertical and horizontal transmission of infection), both of which improved substantially during the period. The potential role of rising living standards in mediating this relationship will be discussed below.

### Survival of the previous sibling and mortality

The mechanisms of the relationship between interval length and mortality are explored further by extending the previous fixed-effects models to include an interaction between the previous child's survival and the length of the preceding interval. Those models were then used to calculate marginal effects of a change in the survival status of the previously born child on the risk of the index child dying. The results can be interpreted as the change in the predicted probability of dying that would occur if the previously born child died at different times with respect to the index child's birth. The results appear in Table 5. To facilitate interpretation, they are presented in three groups: short intervals (<2 years), moderate intervals (2–4 years), and long intervals ( $\geq 4$  years).

Starting with neonatal mortality, the death of a sibling had roughly the same influence on the probability of dying regardless of interval length. Deaths during the pre-gestation period typically reduced the risk of dying slightly, whereas a sibling death during the gestation period led to a slight increase in the probability of neonatal death. There was a very large and positive effect of a sibling death on the index child's risk of dying when it occurred in the postnatal period, however. The death of a sibling during this period led to an increase in the probability of dying of about 0.2, and varied little across interval lengths. In fact, neonates born following longer intervals tended to be more greatly affected by a sibling death in the postnatal period, though this difference was not very large. Overall, there is no clear indication that short intervals are disproportionately associated with any of the proposed mechanisms regarding neonatal mortality.

Turning to post-neonatal mortality, several patterns emerge. First, a sibling death during the index child's prenatal period consistently lowered the child's post-neonatal mortality risk. The negative effect was strongest in the pre-gestation period and weaker but still significant during the gestation period. Interestingly, the magnitude of the protective influence of the sibling's death during the prenatal period was smallest for children born after short intervals and about 60 percent larger for those born after long intervals. If the previous child had died in the index child's postnatal period, there was a strong positive increase in mortality risk for the index child. Once again, the size of this effect depended on the length of the preceding birth interval. The increase in mortality was largest for children born after a short interval, and the effect size decreased as birth intervals lengthened.

**TABLE 5** Marginal effect of the timing of death of the previously born child on the probability of dying for different birth interval lengths

	Neonatal	Post-neonatal	Younger child	Older child
<b>Preceding birth interval &lt;2 years</b>				
$x < -280$ days	-25.2***	-83.0***	23.6***	-0.9
$-280 \leq x < 0$	18.6***	-31.2***	-73.1***	-4.4
$0 \leq x < 28$	193.9***	109.9***	-101.4***	-18.6
$28 \leq x < 365$		171.1***	-99.9***	-3.2
$1 \leq x < 5$ years			54.5***	-60.0***
$5 \leq x < 10$				-106.2***
<b>Preceding birth interval 2–4 years</b>				
$x < -280$ days	-22.9***	-96.8***	-48.9***	-10.0***
$-280 \leq x < 0$	7.6*	-47.6***	-137.1***	-10.5*
$0 \leq x < 28$	171.4***	72.8**	-167.0***	-23.4
$28 \leq x < 365$		130.6***	-156.2***	-14.8**
$1 \leq x < 5$ years			2.1	-111.7***
$5 \leq x < 10$				-81.3***
<b>Preceding birth interval &gt;4 years</b>				
$x < -280$ days	-21.8***	-127.5***	-178.5***	-39.9***
$-280 \leq x < 0$	-7.7	-68.3***	-219.9***	-51.1***
$0 \leq x < 28$	229.4***	-132.3	-251.5	-20.3
$28 \leq x < 365$		35.2***	-169.2***	-41.7***
$1 \leq x < 5$ years			-42.7***	-167.2***
$5 \leq x < 10$				-33.0***

\*p&lt;0.1; \*\*p&lt;0.05; \*\*\*p&lt;0.01.

NOTE: For mortality categories see note to Table 2. Marginal effects calculated based on linear probability models with interaction terms between preceding interval length and mortality of previously born child. Significance indicates the estimated effect is statistically different from the effect of the previous sibling being alive.  $x$  refers to the date of death of the previous child with respect to the age of the index child. Negative integers represent the prenatal period of the index child, and positive integers the postnatal period. ( $x < -280$  days = previous child died before gestation of the index child.) Marginal effects reported per 1000 population to facilitate interpretation.

SOURCE: Roteman Database.

There was no statistically significant association between a sibling's death in the early postnatal period and mortality among index children born after long intervals.

The largest effects of interval length in either direction were on the mortality risks of younger children. Disproportionately large effects of birth spacing on child mortality have also been found in contemporary developing contexts (Ronsmans 1996). The patterns observed for younger child mortality were similar to those of post-neonatal mortality; the mortality-reducing effect of a prenatal sibling death was relatively weak, the mortality-increasing effect of a postnatal sibling death relatively strong. For index children born following a short interval, a prenatal sibling death generally reduced mortality, but this effect was only about one-third as strong as for index children born after long intervals. Interestingly, if a sibling died during the postnatal period, particularly after age one, there was an



increased risk of mortality among children following short intervals, but not following moderate or longer ones. Among children born following longer intervals, a sibling death in the postnatal period actually reduced the risk of mortality.

Finally, mortality risks of older children responded quite differently to the death of the previous child than did mortality at other ages. Sibling deaths primarily influenced the mortality of the index child if they occurred during the postnatal period. For older child mortality, there was no increase in the risk of dying following the death of a sibling in the postnatal period. In fact, the death of a sibling after the birth of the index child always lowered the risk of mortality. There was no clear pattern regarding this relationship and interval length. Index children born after long intervals had the greatest gains in survival following the death of a sibling, but index children born after short intervals also had gains similar to those experienced by children born after moderate intervals.

## Discussion

The results from the preceding analysis provide clues about the mechanisms through which short birth intervals lead to adverse perinatal outcomes. The first evidence of these mechanisms arises from the changing nature of the relationship between interval length and mortality over time. It is clear that the mortality-reducing effect of longer birth intervals disappeared during the study period as mortality declined. Research using cross-sectional data from developing countries has also found that longer birth intervals lower infant and child mortality risks to a greater extent in high-mortality countries (Palloni and Millman 1986). Although it was not the aim of this study to test why this relationship changed, the fact that it did is indicative of the mechanisms linking short intervals to mortality. The weakening of this relationship was likely related to at least one of two trends: improved nutrition or declines in infectious disease. Improvements in nutrition would influence this relationship if it reduced the effects of sibling competition, allowed women to recover from previous pregnancies more quickly, or improved host resistance against infection. During the period of observation, families were able to consume more food for relatively less money. Unsurprisingly, the share spent on food was nearly halved during the course of industrial development in Sweden's capital due to declining or stagnant food prices for much of the period and increasing wages (Molitoris and Dribe 2016a). Thus, the nutritional mechanism may have been weakened during this period.

The decline of infectious disease may also have weakened this relationship if the horizontal transmission hypothesis is the primary mechanism. Like many populations, Stockholm underwent an epidemiological transition during this period. Before the beginning of the twentieth century,

crude death rates were dictated by infectious disease mortality. In particular, a handful of airborne diseases were responsible for the majority of deaths, the deadliest of which were bronchitis, pneumonia, and tuberculosis. By the first decade of the twentieth century, however, deaths from non-infectious conditions such as cancers and circulatory diseases became more prevalent. Mortality from the most lethal infectious diseases had all but vanished by 1926, the end of the Roteman Database's coverage. The link between birth interval length and mortality may thus have been eliminated as mortality from infectious diseases fell if the horizontal transmission mechanism was, in fact, the primary reason for elevated mortality among individuals born after short intervals.

Another crucial piece of evidence presented above concerns how index children's mortality responded to the death of a previous sibling. The death of a sibling during an index child's prenatal period almost universally led to a diminished risk of mortality at most ages independent of the length of the preceding interval. The relationship, however, was typically weaker for those born after short intervals. Postnatal sibling deaths, on the other hand, typically increased the risk of mortality. The analysis showed quite clearly that this mortality-increasing effect was strongest for children born following shorter intervals. When analyzing younger child mortality, only the children born after short intervals were negatively affected by a postnatal sibling death, strongly supporting the horizontal transmission hypothesis in which closely spaced siblings are susceptible to easily transmitted diseases.

The implications of these findings are as follows. First, of the proposed mechanisms linking short intervals to higher risks of dying at young ages, most may still play a role in explaining the relationship, though their roles may be weaker than formerly would have been expected. For example, the mortality-reducing influence of prenatal deaths is indicative of a maternal depletion effect or, possibly, a sibling competition effect in the case of overlap between pregnancy and breastfeeding. Likewise, the mortality increase associated with postnatal sibling deaths is most likely related to the horizontal transmission of disease. It is clear, however, that sibling deaths in the postnatal period were much more significant for the survival prospects of index children, suggesting that the horizontal transmission hypothesis has a dominant role in the discussion of causal mechanisms.

Two hypotheses do not seem consistent with the findings: the vertical transmission of infection and family frailty. The greatest weakness of the present analysis is its inability to identify the vertical transmission mechanism, as it has focused on deaths of siblings, whose health may or may not influence that of mothers. In the analysis of neonatal mortality, there was a positive effect of a sibling death during gestation on mortality, which can be related to vertical transmission if the mother was also infected. But the overall effect size was smaller than the effect of a pre-gestation sibling

death, which influenced mortality in the opposite direction. Furthermore, the analysis was conducted under the assumption that all children were born at full term, which may be less likely for children born following short intervals (Miller 1989). It is therefore possible that the positive association of a prenatal sibling death and neonatal mortality is really driven by premature births whose siblings actually died in the postnatal period. As for the family frailty hypothesis, one would expect a sibling death at any point, pre- or postnatal, to be positively correlated with the index child's mortality. This was not the case. Instead, in all analyses except that pertaining to older child mortality, sibling deaths occurring in the index child's prenatal period tended to reduce that child's probability of dying, while those occurring in the postnatal period tended to increase mortality among index children.

Second, the most noteworthy findings of this study have to do with the relative importance of each effect across interval lengths. Although the present analysis cannot dismiss most of the hypothetical mechanisms, some are clearly more pertinent to the relationship between interval length and mortality. As mentioned above, there was a clear mortality-reducing effect of a sibling death during the index child's prenatal period, which arguably indicates the influence of maternal depletion or sibling competition. When analyzing post-neonatal and younger child mortality, however, these effects were actually about two times *weaker* for index children born after short intervals. For children born following intervals of less than two years, mortality risks were influenced more by the death of a sibling in the postnatal period, indicating that the horizontal transmission mechanism dominated. In fact, for young children there was a mortality-increasing effect of postnatal death only for those born after short intervals. The effects on the mortality of older children were rather different. While there were significant associations between the death of the previous child and the index child's mortality above age five, birth interval length was not a good predictor of mortality at these ages. In general, the death of a previous sibling during the prenatal period had virtually no influence on older child mortality, presumably because considerable selection had occurred by the time a child reached five years old. A sibling's death in the index child's postnatal period, however, reduced the risk of mortality. Interestingly, this association was the weakest for closely spaced children, which is surprising as one would expect that the mortality-reducing benefit of removing a competing sibling would have been the greatest for them. Despite the absence of a relationship between interval length and mortality, the results once again show that, even among older children, those born after shorter intervals were generally disadvantaged.

Finally, the results are not merely a product of unobserved maternal heterogeneity. All models used a within-family design to account for the possibility that mothers who have children after short intervals differ from those who do so after longer intervals for unobserved reasons. Put

differently, these are not simply compositional effects. Endogeneity is a serious issue in the literature that has often, but not always (see Curtis, Diamond, and McDonald 1993; Saha and van Soest 2013; Whitworth and Stephenson 2002; Zenger 1993), been ignored. The results in Figures 3 and 4 showed that the relationship may be even stronger than expected once unobserved heterogeneity is controlled for. Overall, the evidence presented in this study provides strong support for the role of the horizontal transmission mechanism as the link between birth interval length and mortality at young ages.

## Conclusion

This study is the first to show how the relationship between birth interval length and infant and child mortality evolved over time, providing clues to understanding the mechanisms of causality at work. I used variation in the timing of the previous child's death in the index child's prenatal and postnatal periods to better understand the mechanisms influencing this relationship. Finally, the study is one of the few to examine this relationship in a historical population for which data on child mortality are much more abundant, while simultaneously having birth spacing patterns similar to those found in contemporary developing countries.

Although horizontal transmission seems to be the dominant mechanism at work, the results showed that other mechanisms are also relevant. However, this study was unable to test how the strength of these mechanisms varied over time, as sibling deaths became increasingly rare over the period 1878 to 1926. Thus it is unclear whether the relative importance of the mechanisms shifted as the relationship became weaker. This is a critical detail to investigate, given that the level of infant and child mortality was substantially higher in Stockholm during this period than it is in contemporary high-mortality populations.

Our understanding could also be improved by giving more attention to other perinatal outcomes, such as morbidity and biometric measures of both mothers and children. Infant and child mortality in most countries is becoming an increasingly rare event. Apart from the highest-mortality populations in the world, the large majority have infant death rates below 50 per 1,000 live births. It is critical, then, to direct attention to the relationship between birth interval length and other quality-of-life indicators for both women and children, as these may increasingly be the issues of the future.

Lastly, much more work can be done with historical data. Although the data for the present study come from a population at the turn of the twentieth century, the patterns of birth intervals and the relationship between interval length and mortality are nearly identical to those found in contemporary developing countries. Increasingly, individual-level cause-of-death

information is becoming readily accessible in historical population records, and this may provide another way to investigate the mechanisms by which interval length influences perinatal outcomes. Apart from the advantages for studying mortality based on larger numbers of events, many historical datasets are longitudinal as well, allowing researchers to use variation in time to investigate the mechanisms underlying the relationship between birth interval length and child mortality.

## Notes

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1 See Cleland and Sathar 1984; Hobcraft, McDonald, and Rutstein 1985; Koenig et al. 1990; Miller 1989; Miller et al. 1992; Nault, Desjardins, and Légaré 1990; Palloni and Millman 1986; Pebley, Hermalin, and Knodel 1991; Rutstein 2005; Saha and van Soest 2013; Scrimshaw 1978. It has been argued that the optimal birth interval should be between 36 and 59 months (Rutstein 2005), although other studies have found no increase in relative risks of mortality at intervals much shorter than the suggested range (Koenig et al. 1990; Kozuki et al. 2013).

2 Stillbirths and miscarriages were not recorded in the data, but Stockholm's official statistical reports show that the stillbirth rate fluctuated between 20 and 30 per 1,000 total births and declined by only about 5 stillbirths per 1,000 live births throughout the data's coverage (Stockholms Stads Statistiska Kontor 1912, 1924). There is therefore no reason to expect that any observed changes over time are driven by changes in stillbirths.

3 I focus on marital births because birth spacing involving non-marital births introduces problems such as the overrepresentation of out-of-wedlock births at the first parity, which, if followed by a marital

birth, typically would result in a long interval between parities 1 and 2.

4 Admittedly, this operationalization is imperfect. Reverse causality may still be a factor if, for example, an index child was infected, transmitted the disease to the sibling, and then both children died.

5 The Roteman Database does not include information on length of pregnancies, and the definitions of the categories above assume that all births are full-term. This assumption is unrealistic and the impact of this assumption on the study's findings is noted in the discussion section, but for most of the mechanisms being studied this should have little impact on the substantive findings.

6 As expected, censoring tended to become a bigger factor for older children. Among neonates and post-neonates, only 0.3 and 5 percent were not observed until the end of the age span respectively. Among younger children this figure exceeds 20 percent and for older children 40 percent. Admittedly, the high proportions of censored individuals are misleading, as the individuals at risk of dying were more likely to be censored by the ending of the period given the longer time at risk required for full exposure. When the proportions censored are calculated only for cohorts that have the possibility of full exposure, the figures fall to around 13 percent for younger children and 20 percent for older children. The robustness of all of the following models have been checked by estimating them with and without censored individuals and controlling for censoring. These robustness checks do not influence the results in any substantive way.

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